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One Hundred and Forty-second Annual Session

of the

MEDICAL SOCIETY OF DELAWARE

1789-1931

October 13th and 14th, 1931

Hotel Du Pont

Wilmington, Delaware

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Tuesday, October 13th, 1931

Meeting of the House of Delegates

Du Pont Hotel

Club Room

10 A. M.

1. Call to Order.
2. Roll Call.
3. Morris Fishbein, M. D., Editor Journal of the American Medical Association, will address the House.
4. Reading of Minutes of Last Session.
5. Appointment of Committee on Nominations.
6. Reports of Officers:
 - a. President.
 - b. Secretary.
 - c. Treasurer.
 - d. Councilors.
7. Reports of Standing Committees:
 - a. Scientific Work.
 - b. Public Policy and Legislation.

- c. Publication.
- d. Medical Education.
- e. Hospitals.
- f. Necrology.

Reports of Special Committees:

- a. Woman's Auxiliary.
 - b. Health Problems.
 - c. Cancer.
 - d. Syphilis.
 - e. Hospital Survey.
 - f. Library.
8. Reports of Delegates:
 - a. American Medical Association.
 - b. Federation of State Medical Boards.
 - c. Other State Societies.
 9. Unfinished Business.
 10. New Business:
 - a. Resolutions.
 - b. Communications.
 - c. Appropriations.
 - d. Approval of Scientific Program.
 - e. Selection of Meeting Place.
 - f. Miscellaneous.
 11. Adjournment.

**Essayists Taking Part in the Annual Sessions
Are Requested to Make Careful Note of the
Following:**

1. Papers read before the Society become the property of the Society. Each paper shall be deposited with the secretary when read. (Chapter X, Section 3, of the By-Laws).

2. Carbon copies are not accepted. Please turn in originals.

3. Double space all papers and leave plenty of margin, especially on first page.

4. No address or paper before the Society, except those of the President, invited guests, and orators, shall occupy more than twenty minutes in its delivery; and no member shall speak longer than five minutes, nor more than once on any subject, except by unanimous consent.

5. All members must be registered before they can participate in the proceedings and discussions of the general meetings. (Chapter III, Section 1, of the By-Laws).

6. *Essayists will please remember that all papers presented before the Society become the property of the Society and therefore are not to be published or submitted for publication elsewhere than in the DELAWARE STATE MEDICAL JOURNAL.*

Tuesday, October 13th, 1931

General Session

Du Pont Hotel

Club Room

2:00 P. M.

Invocation:

Rev. Frederick Harskarl, Wilmington

Address of Welcome:

Mayor Frank C. Sparks, Wilmington

Presidential Address:

George C. McEfatrick, M. D., Wilmington

Report of House of Delegates.

SCIENTIFIC PAPERS

1. Clinical Results of Radium Therapy in Wilmington General Hospital.

—**Ira Burns, M. D., Wilmington**

2. Radium and Deep Xray Therapy.

—**James Watherwax, M. D., Philadelphia**

3. The Xray Examination of the Sacro-iliac Joint.

—**W. Edward Chamberlain, M. D., Philadelphia**

4. A New Method of Performing the Radical Caesarean Operation (Motion Picture)

—**William Wertenbaker, M. D., Wilmington**

SYNOPSIS—Avoidance of "spill" and risk of peritoneal contamination—control of blood-loss—technique—results in seven cases.

Tuesday, October 13th, 1931

General Public Meeting

Hotel Du Pont

Ball Room

7:30 P. M.

Scientific Motion Pictures by the Petrolagar Laboratories, Inc., Chicago

- No. 1. "Movements of the Alimentary Tract in Experimental Animals."

- No. 2. "The Influence of Drugs on Gastro-Intestinal Motility."

- No. 3. "The Thirteenth International Congress of Physiologists."

8:15 P. M.

- No. 4. "Modern Treatment of Cancer."

—**Joseph C. Bloodgood, M. D., Baltimore**

5. Address:

Morris Fishbein, M. D., Chicago, Editor Journal of the American Medical Association.

Wednesday, October 14th, 1931

General Session

Hotel Du Pont

Club Room

10:00 A. M.

6. Xray Diagnosis of the Chest with Special Reference to Fluid.

—**B. M. Allen, M. D., Wilmington**

SYNOPSIS—The radiologic methods used to demonstrate fluid in the chest, especially when such fluid is so small in quantity or so situated that it is difficult to elicit its presence by physical examinations.

7. Shall the Profession Undertake Control of Specialization in Medicine?

—**Henry O. Reik, M. D., Atlantic City**

8. The Diagnosis and Treatment of Carcinoma of the Large Intestine.

—**W. Wayne Babcock, M. D., Philadelphia**

SYNOPSIS—Peculiarities of carcinomatous growths of the cecum, of the ascending colon, of the transverse colon, of the sigmoid, and of the rectum. Methods of diagnosis. Various operations suggested. Newer methods of avoiding colostomy and of reducing the danger of peritonitis from the operation.

9. Obstetrical Hemorrhage.

—**J. M. H. Rowland, M. D., Baltimore**

SYNOPSIS—Differential diagnosis of hemorrhage in early pregnancy—hemorrhage in the middle third of pregnancy—hemorrhage in the last third of pregnancy—etiology—indication for treatment—intra and post partum hemorrhage.

10. Relations of Delaware Public Health to Indigent Syphilitic Cases?

—**A. C. Jost, M. D., Dover**

Discussion opened by **Talisferro Clark, M. D., U. S. Public Health Service Bureau, Washington, D. C.**

Luncheon

1:00 P. M.

For Members, Guests, and Woman's Auxiliary.

Club Room

2:00 P. M.

11. Address:

—**E. Starr Judd, M. D., Rochester, Minn., President of the American Medical Association.**

12. Post-Encephalitic Disorders and Their Relation to General Practice.

—**Earl D. Bond, M. D., Philadelphia**

SYNOPSIS—An experiment in the re-education of children who have had encephalitis and whose conduct has been disordered, describing the organization of a school in a mental hospital, and the treatment of parallel behavior disorders in other organic brain diseases in convalescence and in general delinquency.

13. Malignant Diseases of the Breast.
Joseph McFarland, M. D., Philadelphia
14. Backache.
—Irvine M. Flinn, Jr., M. D., Wilmington

SYNOPSIS—Admitting the possibility of backache caused by gynecological, genito-urinary and arthritic conditions, this paper is limited to a discussion of purely mechanical manifestations of backache and therefore limited further to mechanical causes of lumbosacral and sacro-iliac pain. Differentiation between the two and causes of each; such as strain, fracture, posture and congenital anomalies. A short general discussion of treatment and xray.

15. The Psychiatric Observation Clinic in Delaware.
—Clyde Bennett, M. D., Farnhurst

SYNOPSIS—A concrete analysis of the inception, development and practical significance of a new and unique plan in the care and consideration of the people of the State of Delaware who seek treatment for mental ailments. A short history of the evolution of psychiatry. The law enacted by the last legislature making possible the establishment of the Psychiatric Observation Clinic is quoted.

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Wednesday, October 14th, 1931

- 9 A. M.—Registration.
9:30 A. M.—Meeting.
Invocation:
Greetings: DR. I. J. MACCOLLUM, *Chairman*
Advisory Committee.
Roll Call.
Report of Secretary.
Report of Treasurer.
President's Report of Philadelphia Convention.

Committee Reports

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Report of Kent County Chairman,
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MRS. W. E. BIRD

Address: "After Two Years,"
DR. G. W. K. FORREST

Address: "The Woman's Auxiliary,"
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Editor Journal of A. M. A.

Addresses:
DR. E. STARR JUDD, *President of A. M. A.*
MRS. WALTER JACKSON FREEMAN,
President-Elect of Woman's Auxiliary
of A. M. A.

MRS. W. WAYNE BABCOCK, Philadelphia
Report of Nominating Committee,
MRS. C. E. WAGNER

Election of Officers.

1:00 P. M.—Luncheon—Members of Auxiliary,
Guests of the Medical Society of Delaware.
Social afternoon.

EXHIBITORS

1. Bausch & Lomb Optical Co.,
Rochester, New York
2. Danforth Drug Co.,
c/o Mr. Thos. Donaldson,
Wilmington, Del.
3. Ho'land Rantos Co., Inc.
156 Fifth Ave., New York, N. Y.
4. Hygeia.....Chicago, Ill.
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GOLDSTEIN'S HEREDO-FAMILIAL ANGIOMATOSIS**(Telangiectasia) With Recurring Familial Hemorrhages****(Rendu-Osler-Weber's Disease)****CASE REPORTS**

HYMAN I. GOLDSTEIN, M. D.

Camden, New Jersey

Heredo-familial angiomas (Goldstein), Osler's Disease, Rendu-Osler-Weber's Disease, or Hereditary (Multiple) Hemorrhagic Telangiectasia with Familial Epistaxis, is a heredo-familial disease of the small blood-vessels and capillaries frequently associated with hemorrhages from the skin and mucous membranes.

Hemorrhages may occur in this disease in one or more members of the affected families. Telangiectases or angiomas usually occur in nearly all the members of the affected families—with or without severe recurrent attacks of nosebleed from early childhood. The telangiectases and the hemorrhages from the nose become more aggravated as the patients grow older—particularly in women at the time of the menopause. It has also been noted that the hemorrhages are worse at or about the time of the menses.

Hemorrhages in this disease may occur from the throat, bronchi, and lungs; from the kidneys and bladder, from the bowel, from the stomach, and from the mucous membrane of the mouth. Subarachnoid and meningeal hemorrhages may also occur from the telangiectases or minute congenital aneurysms.

In the advanced cases the spleen and liver may become enlarged. Splenic enlargement has been reported by Fitz-Hugh, Jr., (Feb., 1931) Giffin (1927), Vulpian (1886), Osler, William Frick (1912), Curschmann (1930), Roles (1928), Schoen (1930), and, perhaps, several others. It appears, too, that in some of these advanced cases an intolerance is developed to blood transfusion. In Fitz-Hugh's recently reported cases, with blood Group IV (0), severe reactions followed blood transfusion.

Interesting instances of large telangiectatic and angiomatous spleens have been reported by Borissowa (1903), Pentmann (1915), Symmers (1921), Langhans (1879), Wassiljeff and Protassewitch (1930), and others, but the cases were not of the typical heredo-familial group here discussed.

Frick's patient, first seen by him in February, 1910, began to develop telangiectatic capillaries on the end of the nose, face, neck, and body during the past fifteen (15) years. At autopsy, the liver was found to be very much enlarged (carcinoma), the spleen was three times the normal size, and dark red in color. The kidneys were both enlarged and congested, showing dilated capillaries were present in the wall of the intestines. Capillary dilatation in the brain were probably also present.

Albert Thierfelder (1873) reported a case of a man with telangiectatic lesions of the submucosa of the bowel, especially in the jejunum and upper ileum and also in the colon and rectum. There were perhaps, seventy (70) angiomas present.

Sequeira, in his book on "Diseases of the Skin," page 579 (1927), in discussing telangiectases, refers to the papers by C. Fox (1908), Hutchison & Oliver (1916), and F. Parkes Weber's papers. He mentions the case of a woman recently under his care, in whom the telangiectases began at the age of forty-one. Her brother and one sister and her paternal aunt suffered from epistaxis, which in two cases had necessitated plugging of the posterior nares.

Strickler (1927), in his book (page 546), makes no mention at all of the heredo-familial type of telangiectases with epistaxis.

Ullmann, on May 8, 1930, reported an additional case (family) of multiple angiomas of the lips and oral mucous membrane, tongue, lower lip, in a man 42 years old. A number of members of the family suffered from nosebleed. (*Dermatologische Wochenschrift* 1931, p. 369, No. 10).

C. W. Bottema reported a family with Osler's Disease before the Holland Dermatologic Association (*Nederl. tijdschr. v. geneesk.* 74: 5560, Nov. 8, 1930).

Hicks and Knox, of the Bellevue Hospital, New York City, reported two cases (one Italian family, and one German family)—an Italian, aged 42, had nosebleed since early childhood, more severe during the past two years. The patient's brother, one sister, father, paternal uncle, and paternal grandmother, all bleed from the nose. The patient's brother and sister each have three children, none of whom bleed. The spleen was not palpable. The liver was enlarged. No syphilis and no alcoholism. Bleeding and clotting time was normal. Blood Wassermann nega-

tive. Blood transfusion was given, but the type of blood is not mentioned. He had bloody stools (hemorrhoids)—proctoscopy, however, showed no telangiectases. They used radium and iodoform gauze pack for the nosebleed. The patient's brother, who was forty-one, had telangiectases on the lips, tongue, and conjunctivae. The second case was a German, male, aged 55 years. He had telangiectases on the lips, tongue, soft palate, left arytenoid, and nail beds. He had nosebleed since early childhood, which increased in frequency and severity as he became older. The patient's mother, son, and a brother had frequent epistaxis. Blood platelets were normal. Blood Wassermann was negative. They (June, 1931), erroneously conclude that there were 19 cases reported in the literature since Goldstein's first paper (*Arch. Internal Medicine*, Jan., 1921), on the subject, making a total of fifty families. They add two additional families. However, they overlooked forty other reported families.

My own study of the subject leads me to believe there are, perhaps, ninety or ninety-five authentic and accepted instances recorded in the entire medical literature of the world—including about five hundred (500) or 550, affected individuals. It is advisable, at this time, to mention H. R. Crocker's case of Multiple Telangiectases (*Atlas of the Diseases of the Skin*, Vol. II, 1896, Plate LXXI, Fig. 1, London). His patient was a girl, aged 14 years, in whom the stellate variety of naevus araneus or spider naevus had been forming from the age of 5 years on the face and forearm. Crocker first saw her in March, 1883, when she was seven years old. The lesions extended over the whole face below the forehead, and more on the left than on the right side, and on the extensor aspect of the forearms, and on the back of the hands. In June, 1890, they extended down the sides of the neck and on to the first phalanges, and there were still some slight involvement of the forehead, chin, and front of the neck. There were minute red points, stellate or loose networks of vessels, and subcutaneous lilac-coloured spots which disappeared on pressure. The parents were well-to-do artisans. Crocker does not mention any other affected members of the family.

J. P. Croes (1847), eighty-five (85) years ago, discussed the subject of "Teleangiectasia congenita;" while M. J. Chelius suggested the treatment of telangiectases with Kreosot (*Med. Ann., Heidelb.*, 1835, i, 93-95).

Among the more recent reported instances of this interesting clinical entity are those reported by Goldstein (1922, 1930, 1931), Fitz-Hugh (1923, 1931), Schwartz (1925), East (1926), Arrak (1925), Emile-Weil (1926), Williams (1926), Foggie (1928), Mekie (1927), McKinstry (1927), Archer (1927), Balph (1927), Mackay and McKenty (1927), Ormsby (1927), Thomson and Mason Lamb (1928), Van Gilse and Postma (1928, 1929), Roles (1928), Flandin and Soulie (1928), Erdheim (1929), Harper (1929), Schoen (1930), Boston (1930), Curschmann (1930), Foldvari (1930), Ullmann (1930), C. W. Bottema (1930), Kenedy (1930), and others.

Leo Kessel, of New York, before the Medical Section of the American Medical Association (June, 1931), at the sessions held in Philadelphia, discussed "Benign Bleeding," and mentioned instances of multiple telangiectases with bronchial, gastric, esophageal, and laryngeal bleeding. In one case of gastric hemorrhages a partial gastrectomy was performed and the patient recovered. Nothing abnormal was found except the presence of many telangiectases.

Jackson, of Camden (1931), informs me that he recently had a case at the local hospital. Kenedy reported a case, a woman aged 80 years, whose mother and sister were similarly affected, before the Ungarische Dermatologische Gesellschaft, at Budapest, October 10, 1930.

D. M. Yazujian, of Trenton (1931), informs me that he has recently seen a case of this disease.

In March, 1931, through the courtesy of Dr. Sussman, of Baltimore, I saw a woman at the Sinai Hospital (in Baltimore), who had had severe hemorrhages for a long time and a number of telangiectases were found present. I was able to trace about 18 or 19 affected members in this family.

Wardner D. Ayer, Syracuse, N. Y. (1931), before the American College of Physicians at the Baltimore meeting, spoke on "Spontaneous Subarachnoid Haemorrhage," and reported 29 cases. He thought the hemorrhages were due to congenital vascular defects—congenital cerebral aneurysms (Circle of Willis). He did not mention hereditary (familial or congenital) telangiectases or angiomas.

The clinical entity discussed here must not be confused with the angiomatous and telangiectatic malformations discussed so thoroughly by Cush-

ing and Bailey, and Arvid Lindau, of Lund, Sweden, Miller, V. Hippel, Kufs (1928), Uiberall (Vienna), and many others. I have reference here, of course, to the cases of retinal angiomatosis as reported by O. Marchesani (1930), P. Junius (1930), G. F. Rochat (1930), Lindau (1930), Miller (1930), and the instances of familial angiomatosis of the retina and cerebellum (Lindau's Syndrome). There are probably seventy-five cases reported in the literature of familial retinal angiomatosis (with cerebellar involvement).

Harvey Cushing and P. Bailey discuss Haemangiomas of cerebellum and retina (Lindau's Disease) and report a case (Arch. Ophthalm. LVII, No. 5, pp. 447-463, 1928, N. Y.) They refer to papers by Scarlett (1925), Fuchs (1882), Th. Leber, Collins (1894), Dzialowski (1900), Coats (1908), Berblinger (1922), Brandt (1921), Rochat (1927), Wohlwill (1927), Shuback (1927), and others.

Hans Ulrik Moller, of the University Eye Clinic, of Copenhagen (Ugeskrift for Laeger, 92: No. 16, pp. 379-384, Apr. 17, 1930), discusses "Familiaer Angiomatosis Retinae et Cerebelli—Lindau's Sygdom." Moller refers to papers by Cushing and Bailey (1928), Lindau, Hippel, Treacher Collins (1894), Meller (1917), Leber, and E. Holm's (1927). Zinsser (1929) reports over sixty (60) cases probably reported in the literature.

G. Eigler discusses "Generalized Angiomatosis" (1930). Schugt (Aug., 1930) reports a rare case of angioma of the ear-drum, auditory canal, face, mouth, tongue, larynx, and pharynx. Heinrich Uiberall (1930), of the Hospital der Israelitischen Kultusgemeinde in Wien, in his paper, "Mit Hauthamangiomen kombinierte Rankenangiome des Gehirns," reviews exhaustively the literature on the subject. He refers to the work of Kufs, F. Parkes Weber, Oppenheim, and the interesting cases reported by Feodoroff and Bogorad, Hubschmann, Emanuel, Steinheil, Cushing, Brushfield, and to the papers by Lindau on "Angiomatosis of the Central Nervous System," and von Hippel on "Angiomatosis Retinae." It is interesting to note that in the case reported by Feodoroff and Bogorad of "Hemangioma of the Brain and Scalp" there was also similar involvement of the spleen and kidneys. Deist (1922) was able to collect from the literature 20 cases of "Rankenangiome des Gehirns" (creeping angiomata).

Arvid Lindau, of Sweden, before The Royal Society of Medicine, (London) Sections of Neurology and Ophthalmology, (Nov. 13, 1930) discussed the subject of "Vascular Tumours of the Brain and Spinal Cord." He says these tumors are uncommon. He mentions Cushing and Bailey's (1928) division into two major groups: (1) angiomatous malformations or capillary telangiectatic malformations, and (2) the haemangio-blastomata or true tumors of blood-vessel elements.

F. Parkes Weber divides the conditions of haemangioma and blood vascular naevi of all types into two main classes:

(1) True neoplastic conditions angio-endotheliomata.

(2) Dilatation and degeneration results—telangiectatic naevi, haemangioma plexiforme, heredo-familial angiomatosis (Goldstein), etc. Hippel's Disease of the Retina and Lindau's Disease, with involvement especially of the cerebellum belong to the first group, whereas, Osler's bleeding telangiectases of the skin and mucous membranes (Rendu-Osler-Weber's Disease), and also plexiform haemangioma of the cerebral meninges, etc., belong to the second group. It is interesting to note that in the encephalon, the second group occur mostly *above* the tentorium cerebelli, whereas, Lindau's Disease (the first group) occurs in the form of haemangio-endothelioma and cyst-formation mostly *below* the tentorium cerebelli (Weber).

Cruveilhier records a case of telangiectasis originating in the brain—(Anatomie Pathologique, Tome II, p. 133-83). Pelletan's case probably also originated in the brain (Clinique Chirurg., Tome II, p. 76).

Zeisler's (1922) patient was a woman aged 22 years who had telangiectasia during pregnancy and disappeared. She had syphilis, but anti-syphilitic treatment had no effect on the lesions. (Arch. Dermat. & Syph. V, 1922, i, P. 781).

John Watson's "Observations on Telangiectasis," read before the New York Medical and Surgical Society, March 2, 1839 (Amer. J. Med. Sciences, Phila., XXIV, No. 47, pages 24-48, May-Aug., 1839) contains reference to Breschet, and others (Memoires de l'Academie Royale de Medecine, Tome III, p. 128). He states that the disease (Telangiectasis) may manifest itself in almost every tissue of the body. Dupuytren says, "It is developed in all parts of the body, but most frequently in the lips, doubtless in con-

sequence of their spongy and vascular structure. It has been met with on the arm, forearm, thigh, scalp, ear, cheek, and organs of generation; in the tissues of the skin, in the muscles, the kidney, the liver, *brain*," etc.

One of Watson's cases (1839) a woman, aged 23 years, had varicosities of the thigh and leg, and a naevus which bled (at the age of 10); she bled occasionally from hemorrhoidal veins and sometimes vomited blood from the stomach. This was one of the most striking cases Watson found on record of general dilatation of the larger veins of a limb connected with a naevus. Watson quotes extensively from the literature and refers to papers by Abernethy, Adams (1835), Cooper (1829), Pelletan, Dupuytren, Bushe, and others. He does not discuss familial cases, nor familial hemorrhages.

In Pentmann's (1915) case of telangiectatic splenomegaly there was profuse proliferation of the endothelial lining of the capillaries in the spleen and liver and the formation of many new capillaries. Cavernomas and angiomas were present in the liver, spleen and bone marrow.

In Symmers' case (1921) of telangiectatic splenomegaly, telangiectatic lesions and dilated capillaries were also present in the skin, lymph-nodes, kidneys, and liver. He refers to somewhat similar cases reported by Borrisowa (1903), and Pentmann (1915). The spleen weighed 1130 gms. (2½ pounds). These instances, however, are not of the true familial variety of hereditary telangiectasia.

Langhans, of Bern (1879, reported a case of a pulsating spleen with cavernous agiomatous tumors, and "metastases" in the liver. The patient was a man aged 30 years. The size of the spleen was: 23 cm. X 15. X 10.5 cm.

"Angiomata" and "Telangiectases" are discussed by Kaposi (Pathologie und Therapie der Hautkrankheiten, pp. 784-790, 5th Ed., Wien, May 1, 1899). Kaposi refers to the "rare generalized" cases reported by Besnier, Kopp (1897) and K. Ullmann (1896), and to the angiokeratomas reported by Wm. Anderson (1898), Mibelli (1889), Pringle (1891), and Dubreuilh.

Pusey, in his book, (1924, 4th Ed. P. 1042), discusses "Multiple Hereditary Telangiectasis with Recurring Hemorrhage," and refers to Goldstein's (1921) paper and his report of 11 members affected in one family, and a collection of 30 families recorded in the literature, and he

refers to Rendu's (1896) first description of this interesting clinical entity.

E. Besnier, L. Brocq, L. Jacquet, (in La Pratique Dermatologique, Tome IV, pp. 447-454, Paris, 1904) discuss "Telangiectases," and refer to papers by Chauffard (1896), Tanturri, Paul Gastou (1894), Vidal (1880), Morrow (1894), Leopold Levi, and Lenoble (1896), Brocq (1897), Leopold Levi and Louis Delherm (1901). They refer to the generalized telangiectasia in a woman reported by Levi and Lenoble (Presse Medicale, 1896, P. 311).

Max Joseph, of Berlin, discusses "Angiomata" (Handbuch der Hautkrankh., Mracek, III, Wien, 1904, PP. 564-579), and includes 54 references to the literature. He refers to Osler (Nov., 1901), Kopp (1897), Lebert (1848), K. Ullmann (1896), Kalischer (1899), C. O. Weber, Gastou (1894), Fulton (June 23, 1900, Jour. A. M. A.), Wolff (1900), Fabiani (Giorn. intern. d. sc. med. XX, p. 1033), Buscke (Aug., 1901), and Virchow (1867). Joseph speaks of Osler's cases of multiple telangiectases of the skin and mucous membranes with epistaxis as *exceedingly rare*—"gehört jedenfalls zu den grossten Seltenheiten."

K. Waltner (Jahrb. f. Kinderh. 106: 307, Aug. 1924) reports a case of "hereditary purpura hemorrhagica." The mother, aged 38, who had suffered from a tendency to purpura since puberty, presented at the seventh month of the pregnancy purpura, metrorrhagia and epistaxis. Examination of the blood confirmed the diagnosis of hemorrhagic thrombasthenia with Werlhof symptoms in the new-born infant as well as in the mother.

C. Julian Lunsford, before the St. Louis Dermatological Society (Oct. 25, 1924), showed a case of multiple angiomas in a girl aged three months. Three other children were normal. No history of similar conditions in the family could be discovered. Dr. Toomey thought this case was similar to those reported by Sir William Osler.

W. Mitchell Stevens, of Cardiff, does not mention Osler's disease or familial epistaxis in his book on "Medical Diagnosis" (London, 1910).

Da Costa, in his "Surgery" (Saunders, Phila., 1931), devotes nearly a page to the subject, but his latest reference to the literature is 1923.

Reference might here be made to the reports by Karl Ullmann, of Vienna: On February 26, 1896, he showed his patient, a woman, with mul-

tipile cavernous angiomata, before the Vienna Dermatological Society. This case was further reported upon by Ullmann in 1899, 1900, and in December, 1930. It was not until his last paper that he made definite reference to this case as being one of Osler's familial and hereditary angiomatoses with hemorrhages. In his monograph of the subject published in the *Festschrift für Moritz Kaposi*, 1900, P. 583, Ullman referred to Rendu's case, reported in October 23, 1896, under the title "Epistaxis Repetees Chez un sujet Porteur de Petits Angiomes cutanes et muqueux." This was the first definite description of nosebleed with telangiectases (heredo-familial), as a *clinical entity*, that I have been able to find in a most thorough study of all the available literature of the world. (*Bull. et mem de la Soc. Med. des hop. de Paris*, 3 serie, Tome XIII, pp. 731-733, Oct. 23, 1896).

Ullmann (1900) refers to reports on "Multiple Angiomata" by Carl Stamm Virchow, Gascoyen, Rokitsky, Billroth, Payne, Thierfelder, Chervinsky, Langhans, Kopp, Hochsinger, Blaschko, Fox, Rendu (1896), Bormann, Schamberg (1896), Du Castel and Hansy, and others.

Ullmann's patient suffered from hemoptysis (1899), due to telangiectases of the bronchial or tracheal mucous membrane. At autopsy, the liver, spleen, and kidneys were found atrophied. The liver showed cavernous angiomata.

Ormsby and Mitchell (1922), before the Chicago Dermatological Society, reported a case of generalized telangiectasia in a woman, aged 33 years, of twenty years' duration. The entire forehead and face were covered with fine telangiectatic areas; telangiectases were present on the neck, chest and arms, fingers and palms. The mucous membrane of the mouth was extensively covered with fine telangiectases. Stokes thought this case belonged to the Osler group. Ormsby in (1922) discussion stated that he had treated a group of cases of Osler's disease several years ago.

Erdheim (Feb., 1929) was able to collect from the literature 55 families with this disease. He reports six cases (who are now alive) in one family, who have frequent attacks of epistaxis with no serious consequences. He also gives reports of five cases deceased, two of which probably died as the result of the severe repeated hemorrhages. He is convinced from his studies of 49 cases that the telangiectatic lesions were

first noticed in 31 cases under the age of 30, and in the other 18 past the age of 30. The lesions seem to become aggravated in many patients in later life.

Fatal hemorrhages in some of these cases were reported by Kelly, Legg, Chiari, Phillips, Gottheil, and others.

Paul (1918) reported the first Australian cases. He reported a woman aged 32 with hereditary angiomata and epistaxis. He traced the disease as far back as the great-grandmother and both her daughters, and grandmother of Paul's patient. Twenty-one members of this family were affected.

Archer (Sept. 17, 1927) reported a case of multiple cavernous angiomata ("of the sweat ducts"); associated with hemiplegia in a man aged 30 years. One brother shows the same telangiectatic lesions. The parents are alive and well. The patient suffered from frequent attacks of bilateral frontal headache. In 1918 he developed a right hemiplegia (at 21 years of age). The attack came on suddenly during the day. Complete recovery took place in two years. In 1922, he had a similar attack in addition to involvement of the left side of the face with loss of speech. He recovered completely from the paralysis.

Barber (Sect. of Dermat. Proc. Royal Society of Med. Oct. 16, 1930, P. 389) reported a case of multiple angiomata in one of twins. The baby girl was ten weeks old, the other child being normal. He also reported a case of a female child, aged 4 years, with angiomata serpiginosum (Nov. 20, 1930, P. 395, Proc.)

Alfred Berliner, of the Krankenhaus Friedrichshain, discussed "Die Teleangiectasien der Blase" (*Deutsche Zeitschrift f. Chirurgie*, LXIV, Chap. XXXI, p. 517, 1902—seventeen (17) references). He also speaks of telangiectases of the skin and mucous membranes. He emphasizes the importance of considering *bladder-telangiectases* as a cause of bloody urine, particularly in young patients. He reports an interesting case, an eleven-year-old school-girl, with asymmetry of face, and persistent recurrent hematuria since the age of 4 years.

Hildebrandt also discusses multiple angiomata of the skin. Virchow saw bladder-telangiectases (*Die krankhaften Geschwulste Bd. III*, p. 453). John Bell (*Principles of Surgery*, P. 341, London, 1826) was probably the oldest observer of "bladder-hemorrhoidal bleeding," even fatal

cases. Nitze (Handbuch der Cystoskopie, Wiesbaden, 1889) also refers to this type of bleeding.

James Miller and D. H. Young, of Queen's University, Kingston, discuss "Essential Haematuria" (Canad. Med. Assoc. Jour., Mar., 1931) in relation to pyelitis of the calyx-papilla angle. They state the term "essential hematuria" has for years been the refuge of the destitute in cases of unexplained and inexplicable kidney bleeding. Angioma of the papilla may explain the greater number of cases of "essential hematuria," according to some observers. Miller and Young report two cases of "essential hematuria": A man, aged 52, with a history of 25 years intermittent hematuria. A woman, aged 38, with hematuria, and constant pain in the region of the kidneys ("pyelitis").

J. H. Bryant (in The Clinical Journal, July 25, 1900, PP. 214-221) published a lecture on "Hematuria" discussing the many causes of hematuria. He mentions Fenwick's (1899) two cases and Debaivieux (1899) paper on "Essential Renal Haematuria," and the cases reported by Sabbatier and Broca, hematuria occurring in apparently perfectly normal kidneys. Bryant's case of "renal epistaxis" or "renal haemophilia," was a woman aged 21 years who suffered nervous shock because a "fire" nearby "frightened her."

Wheeler (New Eng. J. Med. 1928, 198: 123) reports a case of hematuria, two years' duration, in which the kidney and pelvis appeared normal, except for one papilla, the tip of which showed a red, raised, nodule, the size of a pin-head (angioma).

R. Huckel (Zeitschr. f. urol. Chir., 1928, 25: 242), reported three (3) cases, two of which were due to a pin-head size nodule on a papule similar to Wheeler's case, the other to a ruptured arciformis artery.

Senator, in 1891, introduced the term "renal haemophilia." Some of these cases may be due to hemorrhagic telangiectases. Mention should be made of the cases of "familial hematuria" reported by Foggie (1928) and Hurst (1912).

J. W. Hulke, of the Middlesex Hospital (1876) reported a case of general telangiectasis, with extensive involvement. She bled from the bowel and vagina or vulva, according to the mother. She had a naevus on the mons—which caused some bleeding. She died following an attack of erysipelas. (Medico-Chirurgical Trans., PP. 105-

111, (LX, ? 1876 ?), second series, XLII, 1877, London).

Dr. Sinclair (Lancet, II, 1043, Dec. 5, 1885), of the Dundee Royal Infirmary, reported a case of a young leather-cutter, aged 24 years, who had suffered for many years from repeated attacks of epistaxis. On August 19, 1885, he had severe and sudden bleeding from the left nostril, which continued for a number of hours. He died on August 25th. At autopsy a hemorrhage ($\frac{3}{4}$ " long) was found present in the grey matter of the *spinal cord*, disorganizing it, between 6th and 8th dorsal vertebrae.

Osler (1902) discusses chronic splenic anemia—anemia, leucopenia, enlarged spleen—and *recurrent profuse haematemesis*, as the chief symptom. In the later stages, ascites and jaundice are met with in a certain number of cases (Banti's disease).

Emil-Weil and Clerc (1902) report two cases, and review ten other cases, of anemia, with enlarged spleen, with myelocytes and nucleated red cells in the blood. In these cases hemorrhage is a common symptom—two cases had free epistaxis, one hemorrhage from the gums, one retinal hemorrhages, and another metrorrhagia alternating with epistaxis. They call this clinico—pathological entity—"chronic splenomegaly, with anemia, myelemia and hemorrhage."

McIver and Wilson ("Spontaneous Subarachnoid Hemorrhage"—J. A. M. A. 93, No. 2, P. 89, July 13, 1929), state, "there must undoubtedly be some weakness in the blood-vessel wall, which may be either congenital or acquired"—for spontaneous hemorrhage to occur. They discuss spontaneous rupture of the meningeal blood vessels and review the literature. They report 15 cases, varying in ages from 35 to 58 years.

F. Parkes Weber and R. Hellenschmied, in their paper on "Telangiectasia Macularis Eruptiva Perstans" (Brit. Jour. Dermat. and Syph., Aug.-Sept., 1930, XLII, Nos. 8-9, p. 374-382), report a woman (obese), aged 60 years, of phlegmatic temperament, who for 20 or 25 years had a generalized cutaneous macular telangiectasia. The persistent red macules are distributed chiefly over the upper part of the front of the thorax, outer surfaces of the upper limbs and the abdomen.

Interesting Case Reports

Max G.—was a patient at the Lankenau Hospital, Philadelphia, in the service of the late

Doctor Shoemaker. Case No. 3585—1926, Ward II. Other physicians who saw him at the time (Dec., 1926, to Jan. 5, 1927) were Ralph Butler, Stephens, and Rayner. *Provisional Diagnosis was*—Hemophilia (?), Epistaxis, ulceration of left septum. *Examination*—Septum deflected decidedly to right side. *Nose*—large scab on left side of septum and a small one on right side. Bleeds freely after removal of scab on left side. Excoriation on right side (R. Butler, Dec. 28, 1926). *Face* very pale and appears to have reached an advanced state of anemia. *Conjunctivae* very pale. *Mouth*—a small "petechial-like" spot present on lower lip, also some "spots" present upon the buccal mucosa, roof of mouth, and posterior pharyngeal wall. These spots appear to be "very small naevi." Several old teeth present. The remainder being false; tongue pale, but moist; tonsils atrophic. *Heart*—apex beat not visible. Left border 10.5 cm. to left of midsternal line in 5th interspace. *Rhythm*—some sinus arrhythmia, otherwise rhythm is regular. Systolic murmur at apex not transmitted to axilla. Systolic murmur also heard over pulmonary and aortic areas. Muscle sounds of fair tone. *Blood Pressure*—^{S. 118.}_{D. 56} *Abdomen*—no organs palpable. *Extremities*—finger-nail beds pale. Physical examination otherwise negative. Patient made some improvement while at Lankenau Hospital—blood count increased and bleeding decreased. *Blood*—bleeding time, 2½ minutes, coagulation time 3½ minutes. Red blood cells stain poorly. R. B. C.—3,120,000; W. B. C.—9,000; Hb.—50% (12.13. 1926). The patient stated his mother bled from the nose, and died at 46 of diabetes. One brother bleeds from the nose.

Laboratory studies—12.12. 1926. *Urine analysis*—yellow, acid, s. g. 1.021, faint trace of albumin, no sugar, no casts.

At the time of his stay at Lankenau, the true nature of this man's trouble was not recognized.

Max G.—age 62 years, was admitted to Philadelphia General Hospital, October 25, 1928, in the medical service of Dr. David Riesman. Discharged—Dec. 20, 1928. Med. 7298; Ward 5. *Chief complaint*—nosebleed.

Diagnosis—Familial Epistaxis. Notes state he was studied at the University of Pennsylvania Hospital, four times (1910, 1917, 1924, 1926), and at the Lankenau Hospital (1926). He was also a patient at the University Hospital in December, 1929-January, 1930, in Dr. Stengel's service, until his death. He was referred to Dr. Riesman's service as a case of "hemophilia" by Dr. Earl L. Brewer.

History—Had measles. No venereal diseases. Came to U. S. A. in 1902. Did not have nosebleed while in Russia. He was 29 years old when bleeding started. He had pleurisy in 1910 (University Hospital—21 days); typhoid fever in 1917. Three daughters, aged 20, 26, and 32 years—all bleed. Two sisters bled. They died at age 40 and 60 years. Patient has seven children—"living and well"—4 daughters and 3 sons (3 daughters bleed). Has four brothers and one sister "living and well." One brother bled from the nose; the other three brothers did not bleed.

Examination—Several telangiectases on palate, face, lip, and tongue. Spleen not palpable. Liver, from 6th rib to costal margin. Abdomen—negative. No blood from the bowel. No tarry stools. No hematuria. Hemorrhoids in 1924. The notes state that his mother had the same kind of nosebleed; one brother bleeds from the nose; two sisters bled from the nose. His three sons do not bleed; three of his four daughters bleed. Patient's mother died at 46, of "dropsy"; father died at 47 years.

Extremities—varicosities in legs. 11-1-28—Blood count, 2,100,000, R. B. C. 11-6-28, soft systolic murmur over precordium (Riesman). 11-10-28—blood transfusion, 460 c. c. blood. Had slight chilly sensations afterward. 11-12-28

	R. B. C.	W. B. C.	Hb.	Neutro.	Lymph.	L. M.	Trans.	Eosino.
Blood count—12-12-26	3,120,000	9,000	50%	51	39	7	3 —	5
Blood count—12-22-26	3,600,000	7,100	70%	67	20	5	3 —	1
Blood count—1-1-27	4,000,000	7,400	80%	65	25	7	2 —	1
Blood count—1-5-27	4,400,000	6,800	80%	63	27	5	4 —	

—*Blood Press.* S. ¹¹². D. ⁶⁴. Blood transfusion seems not to have relieved him very much. 12-3-28—Dr. Hunter used actual cautery on bleeding points. 12-4-28—Patient feeling better. Has been receiving ultra-violet ray treatments. *Blood calcium* rose from 8.9 mgm. to 9.4 mgm. per 100 c. c. blood, after receiving a number of doses of parathormene. R. B. C.—2,300,000. 12-7-28—actual cautery applied to bleeding points by Dr. Hunter, (who also treated the patient's brother). 12-10-28—R. B. C.—3,620,000. 12-19-28—going home.

Laboratory studies—10-25-28—R. B. C.—2,230,000; W. B. C.—6,000; Hb.—7.5; blood coagulation time—4 minutes; bleeding time—2½ minutes. 10-26-28—blood platelets—230,000; poikilocytosis—two plus; anisocytosis—2 plus; polychromasia; myelocytes. 10-26-28—*Blood chemistry*—urea-nitrogen, .22 mgm. per 100 c. c. blood; sugar, 118. mgm. per 100 c. c. blood. 10-30-28—*Ears*—right ear drum shows sclerotic changes, dull and thickened. Left ear drum shows old healed perforation as result of purulent otitis media. Vertigo is probably circulatory. Several telangiectatic spots on tongue and palate. Ulcerations in Kisselbach's area on both sides. 10-30-28—Fragility test (E. B. C.)—beginning hemolysis .42%, complete hemolysis .24%. 11-5-28—R. B. C., 1,660,000; W. B. C., 5,850; Hb.—6.1; Polys.—89; lymphs.—8; two large trans; 1 Baso.; 1 nucleated red blood cell; anisocytosis; poikilocytosis.

Blood typing—(11-6-28)—Patient Max G., Type IV; Fannie G., Type II; Rose G., Type II. (11-7-28)—Ben. G., Type II.

11-12-28—*Urine*—acid, S. G., 1.020, no albumin, no sugar, few r. b. c., no leucocytes.

11-13-28—R. B. C.—1,350,000; W. B. C.—4,500; Hb.—5.6 mgm.; calcium—8.9 mgm.

11-26-28—Blood platelets—500,000. Bleeding time—3½ minutes. Clotting time—4 minutes.

Blood studies.

	R. B. C.	W. B. C.	Polys.	Lymphs.	Eosin.	Baso.
11-20-28	2,300,000	6,600	55	45	0	0
12-3-28	3,350,000	4,000	72	22	4	2
12-9-28	3,620,000					
12-17-28	3,520,000	6,250				
Dec. 4, 1928	Calcium—9.4 mgm.					

Max G.—Metabolic service of Dr. Orlando Petty. Age 62 years. Philadelphia General Hospital. No. A. 939. Vol. (Series A. 926-949) p. 241.

Diagnosis—Familial Epistaxis, Telangiectasis of turbinates, Diabetes. Second admission. Drs. Crossley, Cippes, Callahan, Matthews, Largey, and Miller saw this patient. Patient was first admitted to the Medical Service of Dr. Riesman, May 8, 1929, and was then transferred to Metabolic, June 21, 1929, discharged July 19, 1929.

Chief Complaint—Nosebleed. Severe anemia. Shipping clerk. Aged 63 years. Born in Russia. H. P. I.—Patient had nosebleed from time to time for almost 30 years. Has had severe hemorrhages since 1910. No pain in the stomach. Perhaps once a year "vomited blood from the stomach," following nosebleed. No hemoptysis. No hematuria. Shortness of breath past six months; typhoid in 1917; pleurisy, 1924. Very little bleeding after tooth extraction. F. H.—Brother, two sisters, grandmother, mother, and three daughters had frequent nosebleed. The other children do not bleed. Wife has high blood-pressure. Patient was a safe-maker for 30 years; for 11 years a teamster, and 11 years a shipping clerk. P. E.—Patient is rather obese; faint yellowish tinge to skin. Pupils regular and equal. React promptly. Conjunctivae quite pale. Artificial teeth. Barrel-shaped emphysematous chest. Heart—Systolic murmur at apex. Abdomen—protuberant abdomen with thick obese abdominal wall. No ascites. Difficult to palpate any underlying organs because of obesity.

May 15, 1929—Blood sugar—200 mgm.

June 21, 1929—Blood sugar—217 mgm.

Blood Studies—

	R. B. C.	W. B. C.	Polys.	Lymphos.
5-24-29	2,500,000			
6-5-29	3,750,000			
6-21-29	4,050,000			
6-25-29	4,150,000	6,500	60	40
6-28-29	4,210,000			
	Hb.—12.4 mgm.			

Sugar

6-21-29—113 mgm., 113 mgm., 98 mgm.; CO₂—48.

6-25-29—100 mgm. sugar. CO₂—41.

6-26—96 mgm. sugar; CO₂—37; Hb.—9.7; P. S. P.—48%, 30%: 78% 2 hours.

5-14-29—*Eyes*—Small hemorrhages. Medic clear. Some yellowish exudate. O. S.—20-50; O. D. 20-70.

6-23—*Blood platelets*—175,000 per cu. mm.; clotting time—3 mins. 3"; bleeding time—1 min. 15".

6-23-29—Oscillometric index—R., A. 4.0; L., 4.0.

7-8-29—*Blood Wassermann*—Negative.

7-1-29—Dr. Hunter advised submucous resection.

7-3-29—Blood Pressure—S. 140, D. 80. Submucous resection performed by Dr. Keenan (10% cocaine plus adrenalin) and this temporarily stopped bleeding.

Electro-cardiographic studies — 5-17-29—No. 12902. The only thing noted is a prolongation of the P—R interval.

6-24-29—No. 13170—The P—R interval is prolonged.

7-5-29—No. 13275—P—R interval is still prolonged and notching of the ventricular complex in Lead I. is noted. Tracing is suggestive of myocardial disease.

7-7-29—*Blood sugars of family*—Cecelia G., —91 mgm. (daughter); Mae F.—124 mgm. (daughter); Mary K.—85 mgm. (daughter); Geo. (Jos.) G.—91 mgm. (son); Doris G.—74 mgm. (granddaughter).

Max G. as reported by Fitz-Hugh, Jr., (Amer. Jour. Med. Scs., Feb., 1931, No. 2, CLXXXI, p. 261), from the University of Pennsylvania Hospital, Medical Services of Dr. Edsall and Dr. Stengel. *Case I.*—A complete summary of the condition of the patient at various periods from December 6, 1910, to the time of his death at the University Hospital, January 18, 1930, and a complete report of the various laboratory studies and examinations are included by Fitz-Hugh in his paper "Splénomegaly and Hepatic Enlargement in Hereditary Hemorrhagic Telangiectasia." Eight females and five males—a total of thirteen members of this family were affected with this heredo-familial disease—(familial epistaxis). Four members of the family are known to have died of the disease—the patient's mother (who married twice), patient's half-sister, the patient, M. G., and a daughter of the patient. The affected son, blood Group O (Type IV Moss), gave blood for transfusion of the patient (Type IV, Moss), on December 26, 1929, with immediate reaction, and with increasing obstructive jaundice until death. The last blood transfusion was given January 17, 1930 (from another relative of identical blood type), followed by sudden decrease in size of the previously enlarged liver, diarrhea, chills, vomiting and death the next day, January 18, 1930. Thus has this patient's medical history (and hospital notes) been

traced for a period of about twenty years. Summary and Comment (Autopsy Report)—as given by Fitz-Hugh—Autopsy (Dr. Custer): Mr. M. G., January 18, 1930. "The skin shows intense jaundice and there are no other external abnormalities." (Note: Telangiectases were present on lips and face, which were obvious and repeatedly described during life.) "All viscera show marked jaundice." Heart essentially negative except for "brown atrophy and fatty degeneration." Small scar of healed tuberculosis at right apex. Moderate pulmonary edema. "No tracheal or bronchial telangiectases." (Note: Bronchoscopy in 1924 had revealed numerous telangiectases—this means therefore that telangiectases had doubtless disappeared from this region as is known to occur occasionally in cutaneous lesions.) The spleen measured (when removed) 19 by 13 by 7 cm. and weighed 660 gm. Its color was slate-gray and its consistency "rubbery." Cut surface was "dark red" and "streaked irregularly with hyaline-like material" which did "not give the color reaction for amyloid." Histologic study of the spleen showed nothing but "chronic hyperplasia; fibrosis" and numerous "areas of hemorrhage." The liver was "mottled" in appearance and was smaller and firmer than normal. It weighed 1300 gm. and measured 23 by 16 by 7 cm. There was no gross ductal obstruction. The gall bladder was thickened and contained "black inspissated bile." Histologic examinations of the liver showed nothing except "toxic hepatitis" and some evidence of "bile capillary obstruction." No ascites. Kidneys showed "cholemic nephrosis." Esophagus was negative except for "petechiae" in mucosa. Stomach and small intestine—normal. The cecum, colon, sigmoid, and rectum showed "homogeneous blue-black discoloration of the mucosa, beginning abruptly at the ilocecal junction." No ulcerations and no telangiectases. Microscopy showed nothing except "melanosis and edema." (Note: Again compare the proctoscopic and biopsy of the rectal mucosa in 1924. The melanosis found at autopsy perhaps represents an end stage of telangiectatic degeneration). Urinary bladder normal. Pancreas and adrenals normal. Bone marrow (mid femur) showed "intense hyperplasia." This is a typical instance of hereditary hemorrhagic telangiectasia presenting in addition the following noteworthy features: (1) Splénomegaly which developed before fifty years of age and

after twenty years of moderately severe recurrent epistaxis and prior to blood transfusion. (2) Hepatic enlargement, which was first noted at age of fifty-eight years (also prior to first transfusion) and which was symptomless until after second blood transfusion. Jaundice and cholemia then appeared and finally, after third transfusion, the picture of acute atrophic toxic hepatitis terminated the story. (3) Increasing intolerance to blood transfusion ending in death within twenty-four hours of the third transfusion. (4) The patient's blood was of Group O (Type IV, Moss). One of his afflicted sons and one non-afflicted son also belonged to the same group. (5) The actual demonstration during life of telangiectases in the bronchial tree and in the rectal mucosa, which could not be demonstrated post-mortem four years later.

The patient I saw, March 25, 1931 (Mrs. Bessie S.), at the Sinai Hospital, Baltimore, with Dr. A. A. Sussman—was a woman, aged 42 years. Attacks of epistaxis began when she started schooling. The hemorrhages have been getting worse during the past three or four years. Lately, the bleeding has been much worse. Examination disclosed a number of telangiectases on the face, tongue, palate, pharynx, and finger-nail-beds. She appeared very anemic, and the skin was a peculiar pale yellow, subicteric tint. She had a palpably enlarged liver and a barely palpable spleen. The patient's daughter, aged 13 years (only child), bleeds occasionally from the nose. Her mother (whom I saw) is similarly affected. The other affected members of her family include her maternal grandfather, great-grandfather, brothers, sisters, aunts, uncles, nieces, and nephews—a total of nineteen affected individuals in the family. I wish to thank Dr. Sussman for the privilege of studying this case.

Felix D., No. Med. 1846. *Epistaxis*. White man, aged 43 years, Philadelphia General Hospital, Medical Service, Dr. S. A. Loewenberg. Admitted November 23, 1927. Discharged November 28, 1927. Orderly. Born in Ireland. In the U. S. A. since 1910.

Chief complaint—Nosebleed. Had the usual diseases of the childhood. No pneumonia. No influenza. Had typhoid fever when a child; no chancre; rheumatism in left knee, 1918. Gonorrhea 14 years ago. Had attacks of nosebleed all his life.

Examination—Face and neck flushed. Well-developed adult. Tonsils cryptic and enlarged.

Blood pressure—^{S. 150.}_{D. 90}. General physical examination is negative. Heart and lungs negative. Abdomen, liver, spleen and kidneys not palpable. No ascites.

11-23-27—Examined by Dr. D. N. Husik.

11-28-27—Etiology of the nosebleed has not been determined.

11-25-27—*Blood*—R. B. C.—4,600,000; W. B. C.—19,900; Hb.—13.8; Polys.—80%; Lymphs.—17%; L. M. and Trans.—3%. *Blood sugar*—126 mgm. *Urea-nitrogen*—15. mgm. *Blood Wassermann*—negative. *Blood calcium*—not done. Had no discomfort from nosebleed. Before the last attack of epistaxis he had headache and dizziness.

In my first paper (January 15, 1921) on the subject, I reported eleven cases in one family. I recently (1931) had the opportunity to see several of these patients—(1) Mrs. Rose W., now 55 years old, has been suffering from very severe nosebleed during the past two years. She is now (June, 1931) very anemic, and very weak. There are many scattered telangiectatic lesions on the face, lips, tongue, etc. In January, 1918, she had a "stroke," from which she made a good recovery after some months. Her condition now, 13½ years later, is very much aggravated, both as to the severity and frequency of the hemorrhages, and the secondary anemia. While her two daughters bleed profusely, her two sons do not bleed, although they have a few scattered telangiectases. She (R. W.) was given blood transfusion (April, 1930) at the Atlantic City Hospital, followed by a rather severe reaction (Blood Type II, Moss).

Mrs. Anna L., now 42 years old, was first examined by me in 1920. She has severe attacks of epistaxis, profuse bleeding during menses, and some rectal bleeding. She is subcyanotic, has clubbed fingers (heart condition), and a number of good-sized angiomas on the cheeks, lips, nose and chin, and fingers. One lesion, on finger-nail-bed, bled profusely recently. Her son, Milton L., now seventeen years old, does not bleed—but has some telangiectases over both ears and face and many small brownish colored spots; some elevated, over the arms, neck and chest. I saw her again June 20, 1931.

Mrs. Elizabeth H.—now aged 45 years, sister of Mrs. A. L. (2) and Mrs. R. W. (1) has a number of scattered telangiectases and frequent nosebleed. Her two sons, Aaron H., aged 21 years, and Marvin H., aged 15 years, and her

daughter, Jeanette H., aged 24 years, all suffer from frequent severe nosebleed. Jeanette H. was admitted to Medical Service II, Mount Sinai Hospital, Philadelphia, October 27, 1925, when 18 years old, because of *epistaxis, anemia*, palpitation on exertion, and vertigo. No. 38502, 10-27-25—11-9-25. She was seen at the hospital by Doctors Rubenstone, Diamond, L. Tuft, Hurwitz, F. Leivy, and Wolffe. The diagnosis was made of "secondary anemia." "Heredo-familial angiomatosis with familial epistaxis" (Goldstein) or "Rendu-Osler-Weber's Disease" was not considered. She has had attacks of epistaxis since early childhood. Menses began at 13 years, ^{28-30.}_{3-5.} Examination—Heart—slightly enlarged to the left; soft blowing systolic murmur heard over entire precordium. Lungs—negative. Spleen and liver not palpable. Septal mucosa "badly diseased" and bleeds on slightest touch. Cryptic tonsils. Blood pressure—^{S. 108.}_{D. 48.} On November 2, 1925, she was given a blood transfusion 355 c. c., which was followed by a marked reaction—chills, urticaria, fever, palpitation, "asthmatic" rales, relieved by injections of adrenalin. 11-5-25—Epistaxis. Blood pressure—^{S. 150.}_{D. 90.} 11-6-25—given sodium cacodylate, 2 grains, intravenously. 11-7, 11-8, 11-9—Calcium lactate, 10 grains, every 4 hours. 10-28-25—coagulation time—4 minutes; bleeding time—3 minutes; blood platelets—340,000. 10-28-25—R. B. C.—2,500,000; W. B. C.—9,400; Hb.—32%; Pmn.—59; S. M.—40; L. M.—1. Slight poikilocytosis, anisocytosis, and polychromaphilia. 10-30-25—Mosenthal Test—Total day—218 c. c. NaCl. 78%; urea.—1%.

	Sahli	R. B. C.	W. B. C.	Pmn.	S. M.	L. M.	Trans.
10-29-25—Hb.	36%	2,420,000	6,800	61	36	2	1
11-3-25	47%	3,410,000					
11-7-25	60%	3,250,000	10,000	80	18	2	

10-28-25—Urine analysis—acid; S. G.—1.018; faint trace albumin; no sugar; some W. B. C.

10-29-25—Urine analysis—acid; S. G.—1.025; no albumin; no sugar; few W. B. C.; no casts. Blood type not given. An aunt, Mrs. Minnie C., was the donor. Mrs. Minnie C., a sister to cases (1), (2), and (4) does not bleed. She has two daughters and a young grandchild who do not bleed. January 12, 1926—under sodium cacodylate intravenously and iron by mouth—Hb.—70%; R. B. C.—3,640,000. Jeanette H. was first examined by me in 1920, at the

age of 13 years. Moderate secondary anemia, with some anisocytosis and poikilocytosis, was present.

(6) Aaron H.—now aged 21 years, I first examined in 1920, when he had a moderate secondary anemia. He was admitted to the Graduate Hospital of the University of Pennsylvania, May 19, 1930. No. 84279, Service of Dr. Carnett, 5-19-30—5-26-30.

Diagnosis—"Anemia," type undetermined. Blood transfusion done by Dr. Wm. Bates. Chief complaint—Pallor, weakness, dyspnoea, nosebleed. The patient had a submucous resection done five (5) years ago. No unusual bleeding. Blood pressure—^{S. 128.}_{D. 90.} Heart—an apical murmur heard, some irregularity. Lungs—negative. Spleen and liver not enlarged. Urine analysis—5-20-30—alkaline; s. g.—1.009; no albumin; no sugar; no R. B. C.; no pus; no casts. Bleeding time—2 minutes. Clotting time—3½ minutes. 5-26-30—R. B. C.—2,400,000; W. B. C.—6,800; Hb.—38%. 5-23-30—R. B. C.—3,030,000; W. B. C.—5,500; Hb.—41%. 5-23-30—Volume index—0.66; Reticulocytes—1%; no normoblasts found; small lymphs—20%; trans.—7%; polym.—73%; blood platelets—176,000; moderate anisocytosis, and poikilocytosis; Blood Type II. 5-23-30—clot retraction—normal; venous coagulation time—5 minutes. 5-22-30—Dr. Steinfield examined the patient and found the spleen barely palpable on deep pressure. He suggested (1) Familial epistaxis with telangiectases; (2) Chronic thrombasthenic purpura (Glanzmann); (3) Secondary anemia or secondary hypoplastic anemia; (4) Primary anemia, as the conditions to be considered. 5-20-30—Transfusion of 300 c. c. of citrated blood. 5-24-30—Dr. Seiberling—septum deflected to the left, causing obstruction. Mucous membrane pale throughout. Middle turbinate hyperplastic bilateral—oozing of blood from antero-lateral surface of same. Strip gauze packing.

Blood chemistry—5-20-30—Blood sugar—91 mgm.; urea-nitrogen—14. mgm. 5-22-30—Blood Wassermann tests—Kolmer, negative; Kahn, negative. 5-24-30—Vanden Berg—Direct, negative; indirect, less than 0.2 mg; Icterus index 4. Fragility test—Haemolysis begins—0.42%; Haemolysis complete—0.30. Dr. Jopson and Dr. Cain performed a partial removal of the nail (right big toe)—on 5-12-31; no unusual bleeding. June 20, 1931, I saw the young

man, who is working for his father, and is feeling pretty good, except for the attacks of nose-bleed.

In November, 1930, Dr. Shmookler saw a patient at the Mt. Sinai Hospital, Philadelphia. Pauline F., age 36, who was admitted to the hospital because of gastric and pulmonary hemorrhages, which were probably due to hypertensive cardiac disease and ruptured vessels rather than to bleeding telangiectases. *Blood pressure*—S. 180. D. 85. *Blood Wassermann*—negative. *Blood urea-nitrogen*—21.6 mgm.; *blood sugar*—97.5 mgm. (No. 60039, 11-15, 11-18-1930).

Differential Diagnosis

The differential diagnosis of Goldstein's Heredo-familial angiomas (angiectasia) with familial hemorrhages or Rendu-Osler-Weber's Disease (Osler's Disease)—must be made from hemophilia, pseudo-hemophilia, the essential and secondary thrombocytopenic and nonthrombocytopenic purpuras, familial thrombasthenia or hereditary hemorrhagic thromboasthenia (Glanzmann), familial purpura, splenic anemia (Banti's Disease), hemogenia, progressive pernicious anemia, malignancy, deficiency diseases (avitaminosis), afibrinogenia, endocarditis, cardiovascular-renal conditions, tuberculosis, hypertensive epistaxis, the various anemias and leukemias, and Lindau's Disease.

Those interested in this clinical entity may refer to Goldstein's papers: *Archiv. Int. Med.*, Jan., 1921; *International Clinics*, Philadelphia, September and December, 1930; *Medical Review of Reviews*, N. Y., April, 1931; and *Arch. Int. Med.*, 1931; *Arch. Dermatiana Syphilis*, 1931.

Newspaper Comment on Code of Ethics

Editorial Note from THE JOURNAL OF THE INDIANA STATE MEDICAL ASSOCIATION: "At the Philadelphia session of the A. M. A. a prominent layman said: 'We do not need more doctors, but more better doctors,' to which he might have added that what we need is more people who will appreciate better doctors and compensate them more adequately for the services rendered." As a matter of fact, sir, he *might* have added that what we also need is a new code of "ethics" which will not prevent a good doctor from telling a layman that one of his fellows of the medical fraternity is actually a dumb, dangerous quack. An innocent child may be at the point of death, improperly treated by an old fogey who chances, somehow, to have picked up an M. D. The condition of that child may be reported to a good doctor. But the good doctor can't take the case and save the child's life until either the perhaps ignorant parents dismiss the blunderer, or somehow contrive to have the blun-

derer call upon the good doctor for consultation. No, the good doctor can't save a life except with the express permission of the quack. The "ethics" of the profession do not permit him to sound warnings against the "respectable" quacks among his fellow-practitioners.—*Fort Wayne News-Sentinel*, July 28, 1931.

Our comment: We do not need a *new* Code of Ethics. Our present code provides rules of conduct that are more beneficial to the public than to the medical profession. Without the Code of Ethics, which is followed by a majority of the reputable physicians, quackery would be more prevalent than it is now. Who is responsible for medical quacks and medical pretenders? The newspapers that advertise them and help fight their battles in the legislature! Medical quackery would die a natural death without the support of the lay press and the politicians who profit directly or indirectly as a result of that support. Incompetent medical men never will be licensed to practice medicine except through the influence of newspapers and politicians who prevent legislation proposed for the purpose of establishing a reasonable standard of qualifications for all those who attempt to treat the sick and suffering. A howl goes up about a "doctor's trust" and "making it hard for the poor boy to enter the medical profession" every time an effort is made to improve the standards of medical practice, and in consequence we have chiropractors and other medical pretenders, most of whom do not possess a common school education, and none of whom know much of anything about the human body either in health or in disease, who are licensed to practice medicine and to impose upon the public. An ailment, diphtheria for instance, curable in the hands of an educated and trained physician, ends disastrously, perhaps fatally, in the hands of a chiropractic pretender. Who makes it possible for these and other quacks to exist and prosper? Newspapers and politicians who sell their souls for a mess of pottage. The medical profession never yet has advocated any medical legislation that has not been of more value to the public than to the medical profession. The Code of Ethics may be hoary with age, but as much may be said of the Ten Commandments. The Code of Ethics includes rules of conduct that are a protection to medical men and public alike, and it would be most unfortunate if the code were abolished or radically changed. If and when the public de-

(Continued on page 177)

EDITORIAL

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VOL. III SEPTEMBER, 1931 No. 9

THE ANNUAL MEETING

An innovation has been made this year in having an evening meeting open to the public. The management of the Du Pont Hotel has kindly let us have the Ball Room for this occasion. Dr. Morris Fishbein, Editor of the Journal of the American Medical Association, will give us one of his enlightening and entertaining talks. He will be with us throughout our session, including attendance at the House of Delegates. Our little Society is fortunate in having both President Judd and Dr. Fishbein as guests and speakers. It is hoped that the physicians of the state will make it their business to inform the people throughout Delaware so that they will have the opportunity of hearing Dr. Fishbein at the open meeting, Tuesday evening, October 13. Dr. Judd, president of the A. M. A., will address us at our meeting Wednesday, the 14th.

Attention is called to the omission by the

County Society of entertainment of the State Society members. This was considered wise at a conjoint meeting of the Scientific Committee and the Committee of Arrangements. New Castle Society established the custom some years ago of entertaining on a rather large scale and the smaller societies felt that they should do likewise. Such procedure throws an unnecessary burden on the smaller groups and it is a good time for the society that started the custom to discontinue it. We think it is wiser to have, instead, such an occasion as we have planned for Tuesday evening. The plan of eliminating the local society from undue expense has been adopted practically everywhere and it is hoped that the members of our Society will appreciate the action that has been taken in this matter. Our State Society still provides one luncheon for its members and guests. We will also have with us the Woman's Auxiliary as our guests at the luncheon.

EDITORIAL NOTES

DEAR DOCTOR:

THE JOURNAL and the Cooperative Medical Advertising Bureau of Chicago maintain a Service Department to answer inquiries from you about pharmaceuticals, surgical instruments and other manufactured products, such as soaps, clothing, automobiles, etc., which you may need in your home, office, sanitarium or hospital. We invite and urge you to use this Service.

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We want THE JOURNAL to serve you.

HOSPITAL FOR CHIROPRACTORS—Governor Murray, of Oklahoma, according to a recent report in the *United States Daily*, has announced that he probably will propose to the next Legislature a bill to construct a state-owned and operated hospital open to all forms of treatment and for use of chiropractors and other non-medical practitioners.

This announcement followed action of the Board of Regents of the University of Oklahoma in refusing to permit chiropractors to the hospital, operated in conjunction with the medical school here. —*New Eng. Jour. of Med.*, Aug. 27, 1931.

What a pity that an up and stepping state like Oklahoma has to tolerate a governor who appears to have absolutely no regard for the health of the commonwealth. Such asininity on the part of Murray, of Oklahoma, makes the conscientiousness of Buck, of Delaware, and Pollard, of Virginia, stand out in bold relief. As a protest

against the tactics of the Governor, Dr. Leroy Long, who has been dean of the Medical School of the University of Oklahoma ever since it started, eighteen years ago, and who really made the school the excellent one it is, resigned his deanship. We fear Murray has done incalculable harm to Oklahoma, but we have no fear that the Oklahoma legislature can be hoodwinked into perpetrating the faux pas that Murray now has in mind.

We note with great regret the death of Dr. Robert J. Ruth, of the Squibb Company, of typhoid fever, in Baltimore, on July 4, 1931. Dr. Ruth was the founder of National Pharmacy Week, and was one of the most forceful and efficient of the many splendid workers who are remaking the pharmaceutical profession. His loss is a great one, and creates a vacancy that will be hard indeed to fill.

On to Wilmington! Do not miss a single meeting of the Annual Session. The program is an excellent one, the roads are perfect, and the doctoring business is so depressed you cannot say you are too busy to come. So come! And bring the wife.

DELAWARE PHARMACEUTICAL SOCIETY

THE PHARMACIST'S PART IN PUBLIC HEALTH SERVICE

Dr. Hugh S. Cumming, Surgeon-General, U. S. Public Health Service, has said that "the usually strategic position and the familiar association of the pharmacy with medical matters in the popular mind places pharmacists in a position to render a material service to the community in connection with public health activities. It is the privilege, as well as the duty, of a pharmacist to co-operate with public health agencies in the dissemination of reliable information concerning the public health, and to assist the constituted public health authorities especially as relates to communicable diseases and the protection of biological products. It is, therefore, evident that a pharmacist should possess information of wider scope on matters pertaining to the public health than is possessed by the average layman.

"Health officers generally have recognized that health education is an important means for pro-

moting public and personal health. Broadly speaking, public health in a given community depends upon the personal health of each individual. To give information on any subject to everyone in a community is a tremendous task, and one that can never be finished. It has no end because new facts are being constantly developed through research and new people are being added to each community through new arrivals and the growth of children to the teachable age. Those who are trying to promote health education for the public have, therefore, the task of imparting an ever-increasing mass of information to an ever-changing population.

"The magnitude of such a task, instead of being a cause for pessimism, should be a challenge to persons interested in the public health to develop a plan whereby each community may feel a sense of responsibility for the important task of health education. Members of the profession of pharmacy can play an important part in the matter of health education.

"The facts for health education are developed by the laboratory workers, those engaged in scientific research of all kinds, the field workers in epidemiology, the vital statisticians, who keep the record of progress, and those clinicians who are close observers of their patients.

"Every person should have a working knowledge of what he should expect in the way of health education from his physician, dentist, pharmacist and nurse, and from the local health organization. In addition to this, he should know what an intelligent and well-organized state department of health may do for the promotion of the health of the citizens of the state, and what may be expected from the Federal Government in the way of health conservation. In order that the pharmacist may measure up to his responsibilities in the matter of the demands for health information, he must, of course, be properly informed with regard to such matters."

In some states the legislators have recognized the importance of pharmacy as a public health service in the provisions which place a pharmacist on the Public Health Board; recently, the example has been followed by several states. It behooves pharmacists to take a deep interest in these matters by seeing that the laws provide for qualified pharmacists in the position, so that the importance of pharmacy may be realized, and as a public health service. In the Colorado

appointment physicians were as intensely interested in the appointment of the pharmacist as pharmacists themselves, and so it should be; they understand the importance of the service and the greater possibilities when all branches of the medical and chemical activities are represented on the board. In Maryland the pharmacists have gained in recognition, because of the service rendered by the officials; it requires a deep interest and knowledge of the problems of this very important service. Quoting Dr. Cumming again:

"The public, generally, is rapidly awakening to the possibilities of preventive medicine, due to the tremendous volume of publicity on health matters that has developed within the past few years. It will be well, however, to remember that the circulation of misinformation by individuals and unrecognized organizations must be avoided. With the development of general interest in public health matters there has sprung up, in certain quarters, an effort on the part of individuals and organizations, for selfish reasons, to disseminate quasi-scientific information that is incorrect, misleading and harmful."

Public health matters are subjects deserving of study by pharmacists and, incidentally, enable them to utilize the information for service and gaining for themselves the good-will of patrons and physicians. The public health broadcasts and bulletins issued by the Bureau of U. S. Public Health Service and State Departments point out ways for being helpful. Public Health Broadcast, entitled "Sanitation of Automobile Tourist Camps" was given to the public on July 1 under the direction of the surgeon-general; we quote a paragraph.

"As early as 1925 it was estimated that in thirty-five states there were more than 3,000 camps attended by over 2,000,000 campers. In 1928, it was estimated that, in California alone, there were 1,400 automobile camps. In other states, particularly those with well-advertised scenic areas, there were probably several thousand others. Investment in camps of all kinds in the State of Maine has been estimated to exceed \$5,000,000. Much has been done within recent years to improve tourist camps, but there is yet much more to do. For example, in one state in 1926 there were 233 camps inspected, and only 104 of these could be approved.

Pharmacists should keep public health bulle-

tins on file for giving information to the public in co-operation with the departments concerned with public health matters; their service can be made of great value, and if this is the outstanding purpose, no one will deny them the right of reasonable remuneration therefrom.

Jour. A. Ph. A., July, 1931.

WOMAN'S AUXILIARY

REPORT OF THE PHILADELPHIA CONVENTION

Though relatively few could attend, yet all our Auxiliary women everywhere are interested in our recent convention, the ninth annual meeting of the Woman's Auxiliary of the American Medical Association in Philadelphia. Because of this interest your national chairman of press and publicity feels that she must give you at least a few items concerning that meeting.

The convention attendance was the largest ever. More than fourteen hundred delegates, members, and guests were present. The program was happily varied with business and recreation.

Activities began Monday, June 8, with a luncheon in honor of the national president, followed by three round-table conferences. These were on: (1) Programs for County Auxiliary Meetings, (2) The Technique and Value of a Committee on Public Relations, (3) History and Archives. These formed a practical, helpful series of discussions.

The convention proper was officially opened by the president, Mrs. J. Newton Hunsberger, at 9 A. M., Tuesday, June 9. Besides much other business, all standing committees reported at this meeting. These were: Organization, Program, Finance, Legislation, Public Relations, Hygeia, Revisions, Press and Publicity, Printing. It is of interest to know we have over 12,000 paid-up members. Income the past year was \$5,338.13 and expenses to April 1 were \$3,087.69.

The program of the Wednesday session embraced, as its outstanding features, the report of state presidents, and the election and introduction of new officers.

The post-convention board meeting was held Thursday morning, and was presided over by the newly installed president, Mrs. A. B. McGlothlan, who outlined her policies for the coming year and announced her committee appointments. Two features of this meeting gave in-

teresting and helpful results. These were the responses to the topic, "What have I gotten out of this convention?" and the discussions incident to opening, "A question and suggestion box."

Not only Pennsylvania, but New Jersey and Delaware assisted in the entertainment provided for this convention. Trips to historic and other points of interest, teas, luncheons and receptions, showed the hospitality and resourcefulness of the splendid convention committee.

Next year the convention will be in New Orleans, in April. Plan now to attend that meeting.

The following paragraphs carry a brief message to you from our president, Mrs. A. B. McGlothlan:

"The reports of the chairmen of the various national committees and of the state presidents indicate unmistakably to the Auxiliary women everywhere that as doctors' wives we have a definite sphere of influence as members of lay women's organizations. As such we may form a strong bond between the medical profession and the lay public.

"Because of this possibility we shall make every effort this year to strengthen our organization both in numbers and in quality of work done.

"The greatest demand made upon us is for the right kind of source material for health programs, and for health program speakers.

"We are attempting to supply this information through a selected packet of literature, assembled by the Bureau of Public Information of the American Medical Association; by leaflets on communicable diseases compiled from the best recent medical literature and approved by a member of our advisory committee appointed for that purpose; by the dissemination of leaflets on 'Some Contributions of Modern Medicine to the World'; by announcement of the American Medical Association radio broadcasts; and by using our best energies to promote the circulation of Hygeia.

"We ask that every doctor's wife read the recommendations concerning Hygeia made to the Woman's Auxiliary by the House of Delegates of the American Medical Association. It is found on page 2116 of the June 20 issue of the Journal of the American Medical Association. Please see that your state and county medical societies also

take notice of this recommendation of the House of Delegates.

"Many Auxiliaries are doing outstanding constructive philanthropic work such as contributing to a medical benevolence fund, assisting in hospital auxiliary work and establishing medical student loan funds.

"We believe that one of the best services we can render to the medical profession is to make our state and national conventions so attractive that great numbers of our women will be enticed to attend and will influence their husbands to come.

"The recent meeting in Philadelphia showed that a convention can serve such a purpose. To this end we are already planning to make the convention in New Orleans the best yet if possible and we herewith invite all the doctors' wives to come and bring their husbands.

"I hope your press and publicity chairman will let me talk with you again. Always read her reports and those in the Bulletin of the American Medical Association. In the bulletin are two pages edited this year, as last, by Mrs. Walter Jackson Freeman, our national president-elect. I commend those pages to you and ask your support to make our departments co-operative, useful, and successful."

MISCELLANEOUS

Poliomyelitis

Of all the infectious diseases of the nervous system, poliomyelitis shows the most definite seasonal prevalence. The disease is always at its height in August, September and October, and this has been the case not only in years when we have had a severe epidemic, such as 1916, but also at other periods when relatively few cases have been reported in Massachusetts. This year, we may expect the same phenomenon to take place and it is the duty of physicians, especially those in general practice, to watch carefully for the disease. So far, the State Department of Public Health has been notified of about sixty cases, thirty-five of which were reported in June. There is no definite indication that the disease is likely to become epidemic. Cases have occurred in many parts of the state. Probably we will have, however, a few more cases this year than have occurred during average years in the past. The type of infection has not been unusual; there have been a number of cerebral and

bulbar cases, with about the average mortality rate.

The State Department of Public Health and the Harvard Infantile Paralysis Commission are well prepared for the disease and every physician should avail himself of the opportunities offered by this unusual service. The commission has been collecting convalescent serum by conducting "bleeding clinics" in various parts of the state. In this way blood is obtained for serum from patients who have already suffered from poliomyelitis. The serum is then bottled and kept ready for use.

Convalescent serum is especially efficacious in the pre-paralytic stage of the disease. Unfortunately, the earliest manifestations are often overlooked by the physician, especially in non-epidemic years. The signs are by no means entirely characteristic, but they indicate a disorder of the central nervous system which justifies further investigation. Given a child with a slightly elevated temperature, who is irritable and restless, who refuses to eat and complains of headache, one should at least consider poliomyelitis, unless there is another obvious cause for the symptoms. One of the earliest signs of poliomyelitis, moreover, is difficulty in sitting up, the patient having a straight, stiff back not easily curved in the natural posture a child assumes when sitting on the bed. Tenderness of the extremities may also be found, with some stiffness of the neck and Kernig's sign. These signs are now considered to be enough to justify lumbar puncture; examination of the spinal fluid, in most cases, will make the diagnosis certain.

If physicians are in doubt in regard to the diagnosis, a special service is now offered by the Harvard Infantile Paralysis Commission to aid in reaching a definite conclusion. Investigators will be sent out by the Commission, supplied with the proper diagnostic equipment, as well as convalescent serum. This service is offered to physicians to aid in the diagnosis and not to supplant them in the treatment of the disease. If, however, the investigator feels, at the time when the lumbar puncture is done, that the diagnosis is reasonably certain, he may, and ought to, give convalescent serum at once. Later injections may be given by the physician in charge.

Poliomyelitis is one of the most terrifying diseases that we have to deal with. It often strikes particularly hard and, if not fatal, it may leave the patient handicapped, so as to preclude the

possibility of earning his living in the future. The after-care of patients with poliomyelitis, therefore, is an important part of the treatment of the disease. Patients should be under the supervision, at least in part, of a physician specially trained. There are a number of centers, moreover, where orthopaedic treatment is available and, fortunate for Massachusetts, a center of this type has been developed at the Children's Hospital in Boston.

Early diagnosis in the pre-paralytic stage, the use of convalescent serum and adequate orthopaedic care when the acute disease is over are the three measures best suited, at present, to combat poliomyelitis. Until we know the cause of the disease, we must rely largely on the use of serum prepared for us by a previous patient, and in its use, much depends on the speed with which an alert physician makes the diagnosis.—Editorial: *New Eng. Jour. of Med.*, July 30, 1931.

Newspaper Comment on Code of Ethics

(Continued from page 172)

s'res to rid itself of quacks and medical incompetents, it can clear the field very quickly if the newspapers will aid in the undertaking, but it takes *newspapers with a conscience* to suppress medical quackery that is profitable to the lay press. The regular medical profession, through its established organizations, at all times is trying to improve the morals and standards of fitness of those who care for the ill. It should be encouraged in its undertakings. Medical quackery criminally preys upon the sick and suffering, more often upon the poor and ignorant who most need protection, but the lay press does little to suppress medical quackery, and it even goes out of its way sometimes to criticize the regular medical profession in its laudable attempts to suppress or wipe out any tendency toward quackery within its own organization. In these days when trickery and chicanery of every kind seem to be profitable, it really is expensive to be good, and we are prepared to take our hats off to the lay publication that has a conscience when it comes to the consideration of suppressing medical quackery.—Editorial: *Jour. Ind. S. M. A.*, August, 1931.

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